1 up

NASA TECHNICAL TRANSLATION NASA TT F-15,826

HEMODYNAMICS IN OBESITY: A BRIEF REVIEW

H. Denolin

Translation of "L'Hemodynamique pulmonaire chez les obeses: une breve revue," Bulletin de Physio-Pathologie Respiratoire, Vol. 8, Sept.-Oct., 1972, pp. 1005-1010

NATIONAL AERONAUTICS AND SPACE ADMINISTRATION
WASHINGTON, D.C. 20546 JULY 1974

STANDARD TITLE PAGE

1. Report No. NASA TT F-15,826	2. Government Accession No.	3. Recipient's Catalog No.
4. Title and Subtitle HEMODYNAMICS IN OBESITY: A BRIEF REVIEW		5. Report Date July 1974
		6. Performing Organization Code
7. Author(s) H. Denolin, Department of Cardiology, Saint-Pierre University Hospital, Brussels, Belgium		8. Performing Organization Report No.
		10. Work Unit No.
9. Performing Organization Name and Address Leo Kanner Associates Redwood City, CA 94063		11. Contract or Grant No. NASw-2481
		13. Type of Report and Period Covered Translation
12. Sponsoring Agency Name and Address National Aeronautics and Space Administration, Washington, D.C. 20546		14. Sponsoring Agency Code

15. Supplementary Notes
Translation of "L'Hémodynamique pulmonaire chez les obèses: une brève revue," Bulletin de Physio-Pathologie Respiratoire, Vol. 8, Sept.-Oct., 1972, pp. 1005-1010.

16. Abstract
Pulmonary arterial hypertension, reflecting left ventricular insufficiency, appears to be common in obese patients without significant alveolar hypoventilation. Isolated right ventricular hypertrophy is rare and chronic cor pulmonale is not usually a complication of simple obesity.

17. Key Words (Selected by Author(s))	18. Distribution Statement
Obesity; pulmonary hemodynamics	Unclassified-Unlimited

19. Security Classif. (of this report)	20. Security Classif. (of this page)	21. No. of Pages	22. Price
Unclassified	Unclassified	9	4.00

HEMODYNAMICS IN OBESITY: A BRIEF REVIEW

H. Denolin
Department of Cardiology, Saint-Pierre University Hospital,
Brussels, Belgium

Obesity is classically included as one of the thoracic dis- /1005*
orders which may result in alveolar hypoventilation and pulmonary arterial hypertension (Bergofsky, 1967).

In reality, although obesity is frequent in occurrence, related hypoventilation is rare. "Simple" obesity should thus be distinguished from obesity with hypoventilation of the Pickwick type, but the data given in the literature are frequently contradictory and do not always permit a distinction between these two conditions. Furthermore, little research has been devoted to pulmonary hemodynamics in cases of obesity not complicated by alveolar hypoventilation. In order to understand the behavior of the pulmonary circulation in obesity without major alveolar hypoventilation, a few general hemodynamic aspects of this clinical condition should first be reviewed (Alexander et al., 1962, 1963, 1964, 1970):

-- Systemic arterial hypertension occurs frequently, in at /1006
least 50% of all cases, but without correlation with the excess weight;

-- The blood volume is increased, with a normal hematocrit test;

-- The cardiac output is increased in proportion to the increase in weight;

---

*Numbers in the margin indicate pagination in the foreign text.

-- The cardiac frequency is normal, implying a greater systolic volume;

-- The arterial-venous difference is normal;

-- The left ventricular work is increased.

However, low values are found for oxygen consumption, blood volume and cardiac output per kilogram of body weight, corresponding to low metabolism and poor irrigation of fatty tissues. The cerebral output is normal (Smith and Alexander, 1960), the renal output is normal, and the splanchnic output is increased. No direct measurements of the rate of blood flow in the adipose tissue are available (Alexander, 1970).

During physical exertion, ventilation and oxygen consumption increase considerably, but the adaptation of the cardiac output is normal: an average of 966 ml/min/100 ml O_2 (Dempsey et al., 1966; Turell et al., 1964).

The pulmonary arterial pressure may behave in three different ways, as in cases of chronic bronchopneumopathy: high pressure at rest and during exertion (25% of cases); high pressure during exertion alone (50% of cases), or normal pressure at rest and during exertion (25% of cases) (Alexander, 1959 and 1964).

The frequent occurrence of normal pulmonary pressure at rest has been emphasized by various investigators. Unfortunately, neither the limits of normality nor the individual values have been given precisely in any of the literature. As early as 1959, Alexander et al. stated that generally the systemic pressure is also high in patients with pulmonary arterial hypertension, although there are some exceptions.

In cases in which it has been possible to measure capillary pressure, an increase in pulmonary arterial pressure at rest or during exertion was always related to a significant increase in venous pressure. Thus in the majority of cases, the major factor in pulmonary hypertension would be an increase in the filling pressure of the left ventricle (Alexander, 1970).

Although a certain degree of desaturation of the arterial blood -- at least at rest -- is frequently observed in these cases, in the Alexander series, which is the only significant one, there /1007 is no correlation between pulmonary pressure on the one hand and arterial oxygen pressure on the other. Moreover, in some cases it has been possible to show that the intraesophageal pressure decreases by a few centimeters of water during exertion. This therefore confirms that, when present, pulmonary arterial hypertension first and foremost reflects a functional change in the left ventricle.

The pulmonary output distribution seems to be normal in cases of "simple" obesity. However, due to disturbances in the ventilation distribution, abnormalities in the ventilation-perfusion ratio may occur which would certainly give rise to the moderate hypoxemia which may be observed in these patients (Dempsey et al., 1966; Holey et al., 1967).

The cardiac volume, estimated on the basis of the transverse diameter of the heart, is increased in most cases, even in subjects with normal systemic pressure (Alexander, 1970). The weight of the heart upon autopsy is high, even in the absence of hypertension or coronary or other lesions. In a series of 12 autopsied cases, Amad et al. (1965) found hypertrophy of the left ventricular wall in nine cases and hypertrophy of both ventricles in only two cases. This was a true muscular hypertrophy and not one due to fatty deposits. Moreover, in the subjects with cardiac insuf-

ficiency, the autopsies consistently showed hypertrophy of both /1008
ventricles and marked symptoms of pulmonary hyperemia. Thus obesity not complicated by severe hypoventilation does not appear to result in true cor pulmonale: right ventricular insufficiency, when it is present and satisfactorily demonstrated (for example, by an increase in venous pressure) is accompanied by changes in the left ventricle and by pulmonary congestion (Alexander et al., 1959 and 1962).

TABLE 1. OBESITY AND HYPOVENTILATION

Pulmonary arterial pressure in cases of obesity with hypoventilation. Influence of hyperventilation (V) or the administration of oxygen ($\dot{O}_2$)

Investigator	$Paco_2$ (mmHg)	Sao_2	PAP (mmHg)	$\dot{Q}$ (l/min)
HACKNEY (1959) a 4 cas			50/30 b à 105/25	
BATES (1971)	62	75	55/16	4,7
+V	40	92	30/16	4,8
GILLAN (1961)		76	80/30	
+ O_2		99	24/8	
HADORN (1958)	60	64	m̄ : 50	
+V	41	81	m̄ : 31	
FRASER (1963)		52	68/36	12,3
+ O_2		98	68/26	

Key: a. 4 cases. b. To.

Although the rate of mortality of obese patients due to cardiovascular problems is high (Plauchu et al., 1970), true chronic cor pulmonale would thus be a cause of death only in exceptional cases. The same would no longer be true of the obesity complicated by severe alveolar hypoventilation which may be observed in some cases. In these cases, pulmonary arterial hypertension becomes frequent, resulting this time from the excess load on the left ventricle, polycythemia, the increase in cardiac output, and especially, the

hypoventilation itself, acidosis, hypoxia and hypercapnia. The role of these factors is well demonstrated by the improvement in hypertension which may be observed under the influence of hyperventilation or oxygenation. In these cases, pulmonary arterial hypertension may be observed with normal "capillary" pressure (Sieker et al., 1955). It should be emphasized, however, that cases of non-reversible hypertension have been reported (Table 1).

In summary, pulmonary arterial hypertension is apparently frequent in cases of obesity without significative alveolar hypoventilation reflecting left ventricular insufficiency. Hypertrophy of the right ventricle alone is rare, and chronic cor pulmonale is not a common complication in non-complicated obesity.

REFERENCES

1. Alexander, J.K., and Dennis, E.W., "Circulatory dynamics in extreme obesity," Circulation 20, 662 (1959).

2. Alexander, J.K., Amad, K.H., and Cole, V.W., "Observations on some clinical features of extreme obesity with particular reference to cardiorespiratory effects," Amer. J. Med. 32, 512 (1962).

3. Alexander, J.K., "Obesity and the circulation," Mod. Concepts cardiovasc. Dis. 32, 799 (1963).

4. Alexander, J.K., "Obesity and cardiac performance," Amer. J. Cardiol. 14, 860 (1964).

5. Alexander, J.K., "The heart and obesity," in: Hurst, J.W., and Logue, R.B., The Heart, MaGraw-Hill, 1970.

6. Amad, K.H., Brennan, J.C., and Alexander, J.K., "The cardiac pathology of chronic exogenous obesity," Circulation 32, 740 (1965).

7. Bates, D.V., Macklem, P.T., and Christie, R.V., Respiratory function in disease, Saunders, 1971.

8. Bergofsky,EE.H., "Cor pulmonale in the syndrome of alveolar hypoventilation," Progr. cardiovasc. Dis. 9, 414 (1967).

9. Dempsey, J.A., Reddan, W., Rankin, J., and Balke, B., "Alveolar arterial gas exchange during muscular work in obesity," J. appl. Physiol. 21, 1807 (1966).

10. Dempsey, J.A., Reddan, W., Balek, B., and Rankin, J., "Work capacity determinants and physiologic cost of weight supported work in obesity," J. appl. Physiol. 21, 1815 (1966).

11. Fraser, R.E., Sproule, B.J., and Dvorkin, J., "Hypoventilation, cyanosis and polycythemia in a thin man," Canad. med. Ass. J. 89, 1178 (1963).

12. Gillam, P.M.S., and Mymin, D., "Hypoventilation and heart disease," Lancet 2, 853 (1961).

13. Hackney, J.D., Crane, M.G., Collier, C.C., Rokaw, S., and Griggs, D.E., "Syndrome of extreme obesity and hypoventilation: studies of etiology," Ann. Int. Med. 51, 541 (1959).

14. Holley, H.S., Milic-Emili, J., Backlake, M.R., and Bates, D.V., "Regional distribution of pulmonary ventilation and perfusion

in obesity," J. Clin. Invest. 46, 475 (1967).

15. Plauchu, M., Pousset, G., de Montgolfier, R., Vemre, B., and Jandot, V., "Vie et mort de l'obese," Lyon Medical 223, 1201 (1970).

16. Sieker, H.O., Estes, E.H., Keger, G.A., and McIntush, H.D., "A cardiopulmonary syndrome associated with extreme obesity," J. clin. Invest. 34, 916 (1955).

17. Smith, W.G., and Alexander, J.K., "Cerebral hemodynamics and oxygen utilization in extreme obesity," Clin. Sci. 20, 33 (1960).

18. Turell, D.J., Austin, R.C., and Alexander, J.K., "Cardio-respiratory response of very obese subjects to treadmill exercise," J. Lab. Clin. Med. 64, 107 (1964).